ANAESTHETIC PROBLEMS OF INTESTINAL OBSTRUCTION IN ADULTS

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SUMMARY

The problems of anaesthetizing ill patients with intestinal obstruction arise from the possibility of aspirating stomach contents, and from attempts to produce adequate abdominal muscular relaxation. The hazards and mechanisms of both vomiting and regurgitation are discussed in detail. On this basis, the rationale of preventing aspiration is described. For the production of adequate abdominal relaxation, the muscle relaxants appear to be the method of choice, despite occasional abnormal responses in these patients. The nature of these responses and methods of rendering them even less common are discussed in detail.

One of the constantly recurring problems which anaesthetists have to face is the patient who requires operation for the relief of intestinal obstruction. Some of these patients are comparatively fit, but others are so gravely ill that they present anaesthetic problems demanding a degree of care and skill which only great experience can provide. These problems have been grouped under two main headings, the aspiration of stomach contents and the provision of adequate surgical access in the abdomen. The purpose of this paper is to discuss these in the light of recent contributions to the literature.

In the pre-operative period there is also a group of problems which, although not the direct concern of the anaesthetist, have an important bearing on his work and they will be mentioned first briefly.

PRE-OPERATIVE PROBLEMS

Fluid and electrolyte loss.

Normally, about 8 litres of digestive juices are secreted every 24 hours, most of which is re-absorbed in the colon. If there is an obstruction proximal to the colon, large volumes of gas (mainly nitrogen) and fluid accumulate above it. Small bowel obstruction presents a more urgent problem than large bowel obstruction because the patient soon becomes gravely ill from dehydration and electrolyte loss. The electrolyte content of the fluid lost depends on whether the small bowel obstruction is high or low. If it is high, a large proportion of the fluid lost is gastric juice (containing H+ ions) and its loss produces a metabolic alkalosis, whereas if the obstruction is low, a much greater loss of pancreatic and intestinal secretions (containing HCO₃ ions) produces a metabolic acidosis. The intestinal secretions also contain potassium in a concentration similar to that of plasma, and its loss has important effects also (vide infra). The changes associated with dehydration and electrolyte loss are not marked in the majority of cases, because the obstruction is usually diagnosed and treated early. In the gravely ill patient, however, these changes have an important bearing on the survival of the patient and on the action of muscle relaxants, and they will be discussed later.

The accumulation of fluid and gas in the bowel causes abdominal distension and the rise in intraluminal pressure impairs the blood supply to the bowel wall. This occurs to an appreciable extent, even without strangulation, and is a cause of the toxaemia seen in intestinal obstruction of any duration.

Strangulation occurs when the blood supply to the bowel wall is cut off. It gives rise to an intensely toxic peritoneal fluid (Barnett and Doyle, 1958; Barnett, 1959a, b; Fine, 1961), the
absorption of which endangers life, and the toxicity of which increases progressively the farther down the alimentary tract the site of strangulation is from the stomach. In addition, great congestion of the bowel is caused because the veins are usually occluded first. This results in the extravasation of plasma or whole blood into the bowel wall and lumen, and the loss may be so great as to require transfusion (Crawford and Nemir, 1960; Savage, 1960).

The replacement of fluid and electrolyte deficiencies is usually undertaken by the surgical team and will not be discussed here. The anaesthetist must be able to recognize such deficiencies, however, and must be able to treat them adequately, for he will have to institute or supervise such treatment himself on occasions.

**Pre-operative medication.**

When the patient's general condition is poor, narcotics, which are depressant, should be given sparingly if at all. Ill and elderly people readily aspirate material into the lower air passages without evoking any protective response. Morphine and its analogues also depress this response in these patients, with the same results (Pontoppidan and Beecher, 1960). If such drugs are required in hypotensive patients, they should not be given hypodermically, but intravenously because of the danger of rapid absorption later when the blood pressure is restored. If atropine is felt to be necessary for dehydrated patients, it is kinder to inject it intravenously at the induction of anaesthesia.

**The timing of the operation.**

Prompt diagnosis and early operation both reduce the mortality of intestinal obstruction. However, a proportion of patients arrive in hospital in very poor general condition, and such patients should not be taken to the operating theatre too soon (Burdeette and Stevens, 1961; Marcus, 1962). There is much to be said for allowing adequate time (2–4 hours) for replacement of fluid and electrolyte deficiencies, reducing abdominal distension, and ensuring that the stomach is kept empty. The aim is to improve the operative risk by getting the patient into the best possible condition for surgery. [The mortality is 12 per cent rising to 50 per cent in patients over 70 years old (Savage, 1960; Scott, 1961).] Once the "drip-and-suck" regime has been instituted the patient's general condition will not deteriorate, provided strangulation is not present. If there is any doubt, however, the patient must be treated as if strangulation is present and operation is mandatory as soon as the patient can tolerate it. [When strangulation has caused gangrene of the small bowel, the mortality is 30 per cent, rising to 90 per cent in patients over 70 years old (Savage, 1960).]

Much work has been done recently on "low flow states" of the peripheral circulation, associated with prolonged hypotension, i.e. intestinal obstruction of some duration. As a result of the low flow rate of blood in the peripheral vasculature red blood cells form aggregates, causing post-capillary occlusion (Gelin, 1961; Gelin and Zederfeldt, 1961; Moore, 1963; Lancet, 1964). This leads to further reductions in tissue perfusion, increasing the metabolic acidosis already caused by hypotension. It leads to micro-infarction of parenchymatous organs such as the kidney where, in addition, the glomeruli may be blocked by aggregations of red blood cells causing oliguria.

Restoration of the blood pressure may not necessarily abolish these changes, which are due to an increase in the viscosity of the blood at low flow rates and a decrease in the suspension stability of the blood. However, the administration of low molecular weight dextran (m.w. 40,000; not the dextran used as a plasma expander) appears to improve the peripheral bloodflow considerably (Bergentz et al., 1961). Metabolic acidosis and "low flow states" have an important bearing on the action of muscle relaxants and will be discussed later.

With the fluid and electrolyte deficits replaced and the peripheral circulation restored, a considerable improvement occurs in the patient's general condition. The extremities are no longer cyanosed and cold but pink and warm; the blood pressure rises to about 100 mm Hg or more and there is reasonable filling of the veins on the back of the hand. As soon as these changes have taken place, the patient should be taken to the operating theatre.
ASPIRATION OF STOMACH CONTENTS IN INTESTINAL OBSTRUCTION

Airway complications, such as aspiration and atelectasis, are supplanting shock, electrolyte imbalance and renal failure as the leading causes of death in surgical patients (Simenstad, Galway and MacClean, 1962). The incidence of aspiration in intestinal obstruction is unknown, but in several series of unselected cases undergoing “elective” surgery, aspiration of detectable, although inconsequential, amounts of stomach contents occurred in about 17 per cent of patients (Weiss, 1950; Culver, Makel and Beecher, 1951; Berson and Adriani, 1954). It must be assumed that in patients with intestinal obstruction this incidence will be much higher unless preventive measures are taken. It is worth noting that approximately 8 per cent of all surgical deaths are due to aspiration of stomach contents in patients with intestinal obstruction (Edwards et al., 1956; Collins, 1960; Clifton and Hotten, 1963).

Clinical.

When aspiration into the lungs occurs in quantity, the patient may die almost immediately from drowning or from profound anoxia following laryngeal spasm, bronchospasm or other airway obstruction. He may also die from reflex cardiac arrest mediated via the vagi, though this calamity is rare. Fortunately, the patient usually survives the immediate crisis. Small amounts of acid material cause coughing and laryngospasm, but larger quantities give rise to an intense bronchospasm and pneumonitis—Mendelson’s syndrome (Mendelson, 1946; Lancet, 1962). The course is acute and critical, and again the patient may die.

Treatment includes restoring the patient’s oxygenation (Morton and Wylie, 1951), placing him in a head-down position, applying tracheobronchial suction and lavage (Bannister and Sarillaro, 1962; Simenstad, Galway and MacClean, 1962), and administration of antibiotics and hydrocortisone (Dines, Baker and Scantland, 1961). The best treatment of the aspiration of stomach contents, however, is prevention.

PREVENTION OF ASPIRATION

Aspiration can be prevented by: emptying the stomach; preventing gastric contents reaching the pharynx, either by vomiting or regurgitation (these are separate and distinct mechanisms); positioning the patient so that aspiration is impossible; sealing off the airway by means of a cuffed endotracheal tube, thereby separating the pharynx from the lower air passages; and avoiding general anaesthesia.

Emptying the Stomach.

A wide-bore oesophageal tube, such as a Levine’s tube, must be passed and the stomach emptied. (A Ryle’s tube is quite inadequate for this purpose.) Even after apparent success, the anaesthetist must proceed on the assumption that the stomach is not completely empty.

Preventing Gastric Contents leaving the Stomach.

Interest is periodically revived in this idea, which was first described 60 years ago (Kausch, 1903). Basically, a tube is passed into the stomach and inflation of a cuff blocks the gastro-oesophageal junction and prevents reflux (Macintosh, 1951; Fisher, 1953; Guiffrida and Bizzari, 1957). However, the oesophagus is a very distensible organ and the pressure from below can dislodge the tube. Consequently, the method has never been widely regarded as reliable.

Preventing Gastric Contents reaching the Pharynx.

(I) Vomiting.

Vomiting may be associated with the colicky abdominal pain of intestinal obstruction, but it usually occurs during induction with an inhalational agent, particularly in patients whose stomachs are not empty. It is a highly integrated act, involving the respiratory centre, the larynx, the abdominal muscles, and the musculature of the stomach and oesophagus (Brown, 1963). It gives ample warning of its onset, and prodromal signs include salivation, swallowing and irregular respiration. In early anaesthesia the patient can still protect his airway by means of coughing or laryngeal spasm. There are three ways in which vomiting can be prevented:

The stomach must be as empty as possible at the actual moment of induction.
General anaesthesia must be deepened as smoothly and as rapidly as possible, so that anaesthesia is deepened to a point where vomiting does not occur. This may be achieved with diethyl ether, after increasing the patient's minute volume by adding carbon dioxide to the inspired mixture (Inkster, 1963). The hyperpnoea induced also helps to prevent vomiting. This technique requires a certain skill and emphasizes the point that these cases must not be anaesthetized by a novice. Some anaesthetists prefer halothane or cyclopropane, adding a liberal proportion of oxygen to the mixture. Others achieve deeper anaesthesia rapidly by injecting a carefully calculated dose of thiopentone. Here again, experience is essential.

The administration of a paralyzing dose of a muscle relaxant as soon as the patient loses consciousness prevents the muscular effort required for vomiting (Wylie, 1963).

The mechanism of regurgitation is quite different from that of vomiting (Atkinson, 1962) and in order to understand it, the factors maintaining gastro-oesophageal competence under normal conditions will be discussed first.

**The competence of the gastro-oesophageal junction.**

The intragastric-oesophageal and pleuro-peritoneal pressure differences. The greater these are, the more likely is regurgitation to occur. The average intragastric pressure required to produce reflux in a paralyzed anaesthetized patient in whom the stomach and oesophagus are in normal relationship is 35 cm H₂O (range, 16-77 cm H₂O) (Greenan, 1961). Clark and Riddoch (1962), using the same technique, found that an average intragastric pressure of 23 cm H₂O caused reflux (range, 13-28 cm H₂O). This compares with an average resting intragastric pressure in anaesthetized subjects of 11 cm H₂O (Roe, 1962).

The gastro-oesophageal angle. This is thought to act mechanically as a flap valve, regurgitation being more likely if the angle is less acute than normal (Marchand, 1954; Sinclair, 1959; Greenan, 1961). It is possible, though, that this factor has been overstressed in the past (Clark and Riddoch, 1962).

The lower end of the oesophagus. Although there is no anatomical structure to account for it, the lower 4 cm of the oesophagus acts physiologically as a tonically contracted sphincter (Atkinson et al., 1957; Fleshler et al., 1958; Botha, 1959; Robson and Welt, 1959; Ingram, Respess and Muller, 1959). In proper functioning is largely independent of the vagal innervation (Greenwood et al., 1962; Clark and Riddoch, 1962) and its tone is reduced by distending the upper oesophagus and by swallowing. This oesophageal segment is only moderately resistant to reflux but it is assisted by mucosal folds, which are invariably present and which plug the conical space where the oesophagus joins the stomach (Botha, 1958). They are held in apposition by the active tone of muscularis mucosae (Dornhorst, Harrison and Pierce, 1954). This tone may be increased by vagotomy or by atropine (Clark and Vane, 1961, Clark and Riddoch, 1962). At all events, both cause an increased resistance to reflux. The mucosal folds, too, are normally only moderately resistant to reflux, but they help to dissipate the intragastric pressure evenly at the gastro-oesophageal junction. In this way the sphincter and the mucosal folds assist each other.

In addition, the oesophagus passes down from a zone of lower pressure to one of higher pressure as it pierces the diaphragm. Since the lower 2-3 cm of the oesophagus are intra-abdominal and its walls are in apposition, this pressure difference further obliterates the oesophageal lumen and tends to extrude the oesophagus up into the thorax. This tendency is resisted by the phrenico-oesophageal ligament (Brown, 1963).

The diaphragm. This is not directly concerned with the competence of the gastro-oesophageal junction (Braasch and Ellis, 1956; Atkinson et al., 1957; Dornhorst et al., 1954; Botha, 1959). The "pinchcock" action of the crura may occur during deep inspiration, but it is precisely in this phase of respiration that reflux may occur (Creamer, 1955). The importance of the diaphragm (in this context) lies in the fact that it maintains the normal relationship of the stomach to the oesophagus and only if this is preserved does the closing mechanism function properly.

Certain patients with a full stomach do not regurgitate as anticipated, whilst others, properly
prepared for elective surgery, do regurgitate. In the second group the patients are often found to have a hiatus hernia of the short oesophagus type (Dinnick, 1961). The history and radiological appearance of the barium swallow are typical of the condition. The patients are often obese, with short thick necks, so that they tend to develop airway obstruction during an inhalational induction. The short oesophagus type of hiatus hernia predisposes to regurgitation by diminishing the gastro-oesophageal angle and by eliminating the mechanical effect of intra-abdominal pressure acting on the lower 2-3 cm of oesophagus. Any airway obstruction enhances these effects (vide infra).

The situation at the upper end of the oesophagus should be noted also. When partially paralyzed either by relaxants or deep anaesthesia, the cricopharyngeal sphincter acts as a valve, allowing an upward passage but no downward passage (O'Mullane, 1954). The oesophagus can hold a considerable volume of fluid and this may appear in the pharynx if the cricopharyngeal sphincter is paralyzed and if an oesophago-pharyngeal pressure difference exists. Once in the pharynx, the fluid must spill over into the larynx.

(II) Regurgitation.

This is a passive decanting of stomach contents, which is much more dangerous than vomiting. It may occur insidiously and even occasionally without the anaesthetist’s knowledge, because there are no prodromal signs.

Factors predisposing to regurgitation.

Any factor which increases the intragastric-oesophageal or pleuro-peritoneal pressure differences, or which increases the gastro-oesophageal angle, predisposes to regurgitation. For example:

1. A raised intragastric pressure.
2. A change in the patient’s posture, so that gravity increases an already raised intragastric pressure (as when a steep head-down tilt is assumed).
3. A reduction in the capacity of the peritoneal cavity, raising the intra-abdominal pressure (as by distending the bowel).
4. Obstructed spontaneous respiration, causing (a) a marked increase in the pleuro-peritoneal pressure difference during attempted inspiration, and (b) overaction of the diaphragm, raising the intragastric pressure.

5. Pulmonary ventilation of a paralyzed patient, prior to endotracheal intubation. The cardia invariably opens when the pharyngeal pressure exceeds 25 cm H₂O, with consequent insufflation of anaesthetic gases into the stomach. If the lungs are ventilated vigorously a high intragastric pressure is soon produced and this, combined with intermittent opening of the cardia during ventilation, makes regurgitation very probable. During ventilation via the nose, the resistance of the nasal air passages produces a lower pharyngeal pressure (and hence less risk of gastric inflation). The pharyngeal pressure (and the risk of regurgitation) is higher during ventilation through the mouth with a pharyngeal airway in position (Ruben and Ruben, 1962).

6. The presence of an intragastric tube renders the gastro-oesophageal junction less competent than normal, and so may predispose to regurgitation.

Prevention of regurgitation.

The measures to be taken are now self-evident.

Reduction of the intragastric pressure. The stomach must be kept empty up to the very moment of induction, when the tube should be withdrawn above the gastro-oesophageal junction. It can be reinserted after the patient has been intubated.

Reduction of the intra-abdominal pressure. Although desirable for several reasons, this is difficult and unsatisfactory. The nitrogen can be “washed out” of the bowel by giving the patient oxygen to breathe (Fine, Banks and Hermanson, 1936; Macintosh, Mushin and Epstein, 1958).*
The fluid in the bowel can be removed by means of a gastro-intestinal tube, for example, a Miller-Abbott tube. Both these procedures are time-consuming, often exceeding 6 hours. The passage of a gastro-intestinal tube is technically difficult, even requiring radiological control, and it diverts attention away from the problem of resuscitating the patient. Clearly each case must be judged on its merits, but as regards the prevention of regurgitation, the reduction of abdominal distension is usually less important than the ability of the anaesthetist to empty the stomach and to keep it empty up to the moment of induction.

**Maintenance of a clear airway.** It is hardly necessary to impress on an anaesthetist embarking on an inhalational induction, the importance of avoiding laryngeal spasm and maintaining a clear airway. Nevertheless, it is clear that the stormier the induction the higher is the incidence of regurgitation (Weiss, 1950; Culver, Makel and Beecher, 1951; Berson and Adriani, 1954). In other words, this calamity becomes less likely with increase in the skill and experience of the anaesthetist.

**Pre-oxygenation.** If endotracheal intubation is to be facilitated by means of a relaxant, the lungs must not be ventilated until the cuffed endotracheal tube is in place. Pre-oxygenation is therefore required in order to tide the apnoeic patient over the period required for laryngoscopy.

**Cricoid pressure.** As soon as the patient loses consciousness, an assistant exerts firm backward pressure on the cricoid cartilage, obliterating the oesophageal lumen. This prevents regurgitated fluids entering the pharynx from below, it prevents anaesthetic gases entering (and distending) the stomach from above, and it facilitates intubation by pushing the larynx posteriorly. Cricoid pressure should not be used to prevent vomiting, however, because the intra-luminal pressure might rupture the oesophagus (Sellick, 1961).

**Positioning the Patient to render Aspiration impossible.**

**Head-down tilt.**

In this position the patient may vomit or regurgitate, but gastric contents finding their way into the pharynx tend to flow out of the mouth, rather than flow against gravity up into the larynx.

There are, however, several difficulties. Although aspiration is prevented, the larynx may be submerged so that the pharynx must be cleared rapidly if regurgitation occurs. Furthermore, gravity increases the intragastric pressure and the abdominal contents come to lie on the diaphragm. A greater respiratory effort is required, increasing the pleuro-peritoneal pressure difference. Both these factors predispose to regurgitation. However, the risk is not great and can be minimized by ensuring that the tilt is not too steep.

The head-down tilt is sometimes combined with the left lateral position, as an extra precaution. It has been shown that to avoid aspiration in this posture a tilt of at least 20 degrees is required. This is very steep. In addition there must be a clear egress from the mouth to allow any fluid reaching the pharynx to escape; furthermore, the patient must be endentulous so that a cuffed endotracheal tube can be inserted without delay (Elliott, 1963). A head-down tilt is usually combined with an inhalational induction of anaesthesia.

**Head-up tilt.**

This posture is usually adopted when intravenous induction of anaesthesia is contemplated (Snow and Nunn, 1959). Vomiting is prevented by use of a muscle relaxant and this leaves only the problem of regurgitation to be solved. The patient is tilted so that the larynx is at such a vertical height above the gastro-oesophageal junction that the intra-gastric pressure is unlikely to exceed this hydrostatic pressure. In these circumstances, gastric contents, even if forced into the oesophagus, will not reach the larynx.

There are two serious drawbacks to this manoeuvre. The intragastric pressure cannot be estimated (see range of pressures on page 441) and so the required degree of head-up tilt cannot be determined. Should stomach contents reach the pharynx, then the patient is positioned so that aspiration is bound to occur. Further, gravely ill patients become markedly hypotensive in the head-up position, although this can be minimized by adjusting the operating table to form a "V" or "N" shape, and by maintaining the position.
for only a minute or so during the actual induction of anaesthesia.

Hypotension may also be produced even by a small dose of thiopentone, and many anaesthetists prefer to induce anaesthesia with a suitable mixture of cyclopropane (30-40 per cent) in oxygen, when the patient is in this position.

**Sealing off the Airway.**

Aspiration of stomach contents from the pharynx is prevented by sealing off the airway. This is achieved by passing a cuffed endotracheal tube and the patient is in considerable danger from the moment consciousness is lost until this has been done. All methods of inducing general anaesthesia in intestinal obstruction rely on the speedy insertion of such a tube, and the anaesthetist must ensure pre-operatively that the patient can, in fact, be intubated. He should check on inconveniently spaced teeth, arthritis of the cervical spine or temporomandibular joint, and on whether the patient has a short, thick neck, or not.

In patients in whom endotracheal intubation is likely to be exceedingly difficult, a safer alternative is to pass a cuffed endotracheal tube before the patient is anaesthetized, so that he can protect his own airway until it has been sealed off. Unfortunately, it is necessary to depress the protective laryngeal reflexes with local analgesia, in order to intubate a conscious patient, so that much of the safety of the method is thereby lost.

**Avoiding General Anaesthesia.**

At one time aspiration was so feared that it was considered safer for the patient to remain conscious and in possession of the cough reflex by employing conduction anaesthesia. The technique was thought to avoid any deterioration in the patient’s general condition due to “toxic” inhalational agents, and it provided good relaxation. There are few valid reasons for employing conduction anaesthesia today.

Subarachnoid and epidural blocks may themselves cause nausea and vomiting (Moore, 1955) and patients have died from aspiration of vomitus whilst a subarachnoid block was being performed in the sitting position. The retention of consciousness does not necessarily prevent aspiration (Clark, 1963). Further, if the upper abdominal muscles are affected by a subarachnoid block, the patient cannot cough effectively although he may still be able to inspire deeply (Egbert, Tamersoy and Deas, 1961). This contrasts with a high epidural block which, by reducing the capacity for forced expiration only slightly, preserves an effective cough (Moir, 1963).

Conduction anaesthesia may cause a deterioration in the patient’s general condition by producing hypotension. In the elderly this occurs because the closure of intervertebral foramina renders an epidural block unpredictable and more extensive than intended (Mostert, 1960). A similar result is obtained by administering a subarachnoid block to a dehydrated patient (Lee, 1959). Hypotension may also occur as a result of handling the bowel at operation, unless the surgeon can block the vagi (para-oesophageal). This may be impracticable if he is working in the lower abdomen.

A field block is unlikely to permit the return of distended bowel to the peritoneal cavity, so that either a subarachnoid or an epidural block is necessary to provide adequate relaxation. Both will produce hypotension if the required number of segments are blocked. Both may reduce the respiratory tidal volume, and if the patient is hypoventilating from any other cause, such as pre-existing respiratory disease or splinting of the diaphragm by abdominal distension, he will become hypoxic. The combination of hypotension and hypoxia is lethal and must be avoided (Mushin, 1942; Edwards et al., 1956; Bonica et al., 1957; Lund, Cwik and Quinn, 1961). In fact, ill patients with intestinal obstruction fare better with a properly administered general anaesthetic (Bonica, 1958).

To summarize, it appears to the writer that both thiopentone and a head-up tilt are inherently dangerous in these patients, and that probably the greatest safety here lies in an inhalational induction with the patient in the head-down position. Aspiration is very unlikely in such a posture and some reflex activity is preserved almost into the stage of surgical anaesthesia. Furthermore, with inhalational anaesthesia from the start, the anaesthetist can observe and control the patient’s progress. This is not the case with an intravenous induction.
It is not always appreciated that the patient is in danger of aspirating gastric contents in the immediate postoperative period also. Consequently, the anaesthetic technique should allow a rapid return of consciousness with protective reflexes and it must avoid, as far as possible, agents likely to cause postoperative vomiting. The patient should recover in the lateral (tonsil) position under skilled supervision as in a recovery room. Further consideration of the postoperative period leads to the other major problem, which is that the effect of any agent used to provide good surgical relaxation may persist into the postoperative period, causing hypoxia and inactive protective reflexes, and thereby endangering the patient's life.

**THE PROBLEM OF SURGICAL ACCESS**

Inadequate muscular relaxation may render the surgical procedure a near-impossibility in a patient with a distended bowel. It is, therefore, in the patient's best interests for the anaesthetist to provide adequate relaxation of the anterior abdominal wall. Such relaxation can be achieved by the use of conduction anaesthesia, inhalational agents or muscle relaxants.

Conduction anaesthesia, although capable of providing good relaxation, is contraindicated in cases of intestinal obstruction as already stated. The use of an inhalational agent ensures that when the agent is exhaled at the end of the operation the tissue concentration falls and muscle tone and power return. Unfortunately, the depth of anaesthesia required in order to produce the desired relaxation often causes respiratory depression and results in cardiovascular depression with hypotension. Recovery of both consciousness and protective reflexes in the immediate postoperative period is delayed. In view of these drawbacks, inhalational agents have only a limited place in providing adequate relaxation in cases of intestinal obstruction as already stated.

The anaesthetist's armamentarium still contains the muscle relaxants, however. They possess several advantages in that the dose used can be controlled accurately, only a light general anaesthetic is required, and their action is readily reversed after operation, so that the patient awakens quickly and is able to protect his airway.

**The Relaxants.**

**Depolarizing agents—suxamethonium.**

This provides optimal relaxation so that the trachea can be intubated rapidly, minimizing the time that the airway is at risk. However, suxamethonium occasionally increases the intragastric pressure (Roe, 1962; Andersen, 1962), due to contraction of the abdominal musculature and abnormal movements, caused by muscle fasciculation. This is yet another reason for ensuring that the stomach is empty at the moment of induction.

If the anaesthetist intends to use a longer acting non-depolarizing relaxant during the operation, it is wise to wait until the effect of the suxamethonium is wearing off before giving the non-depolarizing drug, otherwise a prolonged neuromuscular block may follow. The practice of administering two different types of relaxants in the same anaesthetic sequence is better avoided, however, especially in cases of intestinal obstruction.

If suxamethonium is used throughout a laparotomy, the likelihood of some neuromuscular block persisting into the postoperative period depends on the method of administration, the total dosage and the rate of hydrolysis. The production of continuous adequate relaxation is difficult using intermittent doses of suxamethonium, though the method ensures that the previous dose is hydrolyzed before the next is given. The rate of hydrolysis depends on the level of the plasma cholinesterase (pseudocholinesterase), but the plasma level is usually normal in intestinal obstruction, provided there is little inflammation or sepsis (Vorhaus and Kark, 1953). Recently, suxamethonium has been used in reduced dosage, its rate of hydrolysis being slowed by tetrahydroaminacrine, an anticholinesterase (Barrow and Smethurst, 1963; Kenton, 1963). The safety of this interesting approach remains to be seen, but the initial reports in ill patients with obstruction are encouraging. Catabolic protein loss is maximal about one week after laparotomy (Wilkinson et al., 1950) and the plasma cholinesterase is around the lower level of normal at this time (Burnett, 1960). Should the bowel become obstructed, due to adhesions, about a week after laparotomy, the...
anaesthetist should be wary of using suxamethonium during the second operation.

Non-depolarizing agents—tubocurarine and gallamine.

Patients with fluid and electrolyte deficiencies usually have an increased sensitivity to muscle relaxants, especially of the non-depolarizing type. This is probably due to an increase in the ratio of intracellular to extracellular potassium, a reduction in the volume of the extracellular fluid, and a reduction in the volume of urine excreted (Foldes, 1957a, 1960).

The acute loss of extracellular potassium in the intestinal secretions is aggravated by the lack of potassium intake from nausea and vomiting. In severe dehydration, intracellular potassium is lost also. Although oliguria tends to maintain the ratio of intracellular to extracellular potassium ($K_I / K_O$), re-hydration of the patient with saline only will increase the ratio. This causes the cell membrane to be hyperpolarized and refractory to depolarization, so that the myoneural junction becomes more susceptible to non-depolarizing relaxants (Feldman, 1963). Consequently, potassium replacement should be considered in these patients (Taylor, 1963).

After intravenous injection of a relaxant, its plasma level and its concentration at the myoneural junction come into equilibrium, and the maintenance of the neuromuscular block depends on the plasma level. In turn this depends on the redistribution of the relaxant into the extracellular fluid. If the volume of extracellular fluid is reduced, a particular dose of relaxant results in a higher plasma level than usual.

The plasma level of a relaxant also depends on the rate of excretion in the urine. If the volume of urine being excreted is small, then the plasma level will fall slowly and a given dose of relaxant will have a longer effect than normal. Gallamine is completely, and tubocurarine partly, excreted in the urine.

There are several possible causes for persistence of the action of non-polarizing agents into the postoperative period.

Overdosage. The anaesthetist should not administer such a dose of gallamine or tubocurarine that the effect cannot be reversed by a reasonable dose of neostigmine at the end of operation. To prevent overdosage it is essential that both the anaesthetist and the surgeon differentiate between any difficulty due to distended bowel on the one hand, and inadequate muscular relaxation on the other.

A poor peripheral bloodflow. Muscle bloodflow is very important in the production of a neuromuscular block (Churchill-Davidson and Richardson, 1952). Cases of intestinal obstruction often have a poor peripheral circulation ("low flow state"), and this slows down considerably both the onset of paralysis and the subsequent recovery of normal muscle power and tone. The neuromuscular block may be prolonged in these patients. Such a situation may be avoided by pre-operative restoration of the blood volume and hence the blood pressure, but the peripheral bloodflow is probably best improved by the administration, in addition, of low molecular weight dextran (m.w. 40,000).

Intraperitoneal antibiotics. The antibiotics streptomycin, neomycin, polymixin and kanamycin all have curare-like properties (Sabawala and Dillon, 1959). They lower the blood calcium and thus affect the membrane potential of the muscle endplates (Corrado, 1963). Cases have been reported in which large intraperitoneal doses of these antibiotics reinforced the residual neuromuscular block after the use of non-depolarizing relaxants to such an extent that respiratory insufficiency resulted postoperatively (Pridgen, 1956; Webber, 1957). Such antibiotics are frequently given by this route at the end of an operation for intestinal obstruction, especially if strangulation is present, so the anaesthetist should be on his guard. A slow intravenous injection of 10 per cent calcium gluconate reverses the block and is more effective than neostigmine (Pittinger, Long and Miller, 1958; Jones, 1959; Pandey, Kumar and Badola, 1964).

Re-curarization. Although rarely encountered in practice, it is (at least theoretically) possible for a patient to become re-curarized. There are three possible reasons for this. Firstly, urinary excretion is much reduced in dehydrated or hypotensive patients, and relaxants which are eliminated by the kidneys may still be acting...
after the effect of neostigmine has worn off (Jenkins, 1961). Secondly, in the postoperative period water passes from the extracellular fluid into the intracellular fluid. This increases the concentration of relaxant at the neuromuscular junction (Foldes, 1957b). Thirdly, if a patient whose \( \frac{K_f}{K_o} \) ratio is only being maintained by oliguria (vide supra) is given a non-depolarizing relaxant before being re-hydrated with saline only, then the subsequent increase in the \( \frac{K_f}{K_o} \) ratio will intensify any myoneural block still present. The patient will thus appear to have become recurarized (Feldman, 1959, 1963). These effects should seldom be seen provided that overdosage with relaxants is avoided, and that fluid and electrolyte deficiencies are corrected pre-operatively.

Neostigmine-resistant curarization. The term is unsatisfactory because non-depolarizing relaxants have never been directly implicated. The syndrome was reported in six ill elderly and dilapidated patients, suffering from intestinal obstruction (Hunter, 1956). Anaesthesia was induced in all patients with thiopentone and, after intubation by means of suxamethonium, was maintained using nitrous oxide, oxygen and pethidine, with muscular relaxation obtained using tubocurarine or gallamine after the effects of suxamethonium had worn off. Postoperatively, they were unrousable and their paralysis was never completely reversed clinically. The patient had tracheal tug, peripheral cyanosis and inadequate tidal volumes. The fundamental point is that they all died, not from respiratory insufficiency but from circulatory failure. In spite of a long and involved correspondence in the journals, the aetiology of the condition remained obscure until recently. It has now been suggested by Brooks and Feldman (1962), that the underlying cause is a metabolic acidosis. They reported five cases, presenting the typical clinical picture with apparent “curarization” (e.g. tracheal tug and inadequate respirations), cardiovascular depression with hypotension and peripheral cyanosis, and a fatal outcome. All these cases were found to have a marked metabolic acidosis, with a low arterial blood pH and a low plasma bicarbonate. Subsequently, a similar group of patients, in whom the acidosis was treated, did not die.

Patients with intestinal obstruction often present for surgery with a base deficit and with a “low flow state” of the peripheral circulation. Both produce metabolic acidosis, which is aggravated by anaesthesia and by any hypoxia or hypotension or by massive transfusions of stored blood. Metabolic acidosis causes depression of the central nervous system, producing drowsiness or unconsciousness; depression of the respiratory centre, with inadequate ventilation producing hypercarbia and hypoxia; and depression of the cardiovascular system (Bunker, 1962). Tracheal tug is also thought to be due to acidosis (Scurr and Feldman, 1962). In severe acidosis (pH<7.0), the ventricular contractile force of the heart weakens (Thrower, Darby and Aldringer, 1961), and this causes hypotension and peripheral cyanosis. All these effects may be ascribed to dehydration or blood loss, and metabolic acidosis is not suspected.

The blood pH should be restored to normal by means of a buffer, such as a solution of sodium bicarbonate [approximately 1.3 m.equiv/kg (Brooks and Feldman, 1962), approximately 6 m.equiv/kg (Thrower, Darby and Aldringer, 1961)], or a new organic buffer such as THAM [trishydroxymethylaminomethane (Nahas, 1959)]. This procedure should be carried out under biochemical control whenever possible. The administration of a buffer does not of itself reverse the state of “shock”, but it allows time for the usual methods of resuscitation to become effective.

It is not known whether this explanation covers all cases of “neostigmine-resistant curarization”, but it seems that metabolic acidosis is one cause of the syndrome. Consequently, the pre-operative correction of fluid and electrolyte deficiencies should prevent its occurrence postoperatively.

The causes of persistent relaxant action have been emphasized, for reasons already mentioned, because of its seriousness and because its incidence is somewhat increased in cases of intestinal obstruction. It must be remembered, however, that its overall incidence is low and it would be still rarer if the pre-operative preparation of the patient and the administration of relaxants were invariably carried out correctly. With these
provisos, the writer considers the use of muscle relaxants to be the method of choice for providing adequate surgical access in cases of intestinal obstruction.

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REFERENCES


The problems which arise when anaesthetizing patients suffering from various disorders of the endocrine glands are now well known to expert anaesthetists. The problems of the diabetic patient are given refreshingly reasonable and logical treatment; while the account of the physiology of the adrenergic mechanism, and the sympathetic nervous system in general, will be an eye-opener to the beginner. Throughout the book the editor sprinkles his own pithy and always helpful comments. Although many of the contributors are from the University of Texas, South Western Medical School, Dr. Jenkins has not hesitated to range widely in the United States for his colleagues and even to draw on five British anaesthetists as well. Not the least rewarding aspect of this book is to see the different ways in which a patient and his problems can be regarded by various experts. That they sometimes appear to have little in common is more likely to indicate that they are all right, rather than the reverse, for each sees the problem from his own standpoint. Books like this serve an excellent model for other editors who have the task of covering a localized area of anaesthesia with a group of experts.